WEST Search History

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DATE: Thursday, February 23, 2006

Hide?	Set Name	Query	Hit Count
		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR	
☐	L1	blood near group near binding	15
☐	L2	hops or hop-s or hops	41251
☐	L3	L2 and (helicobacter or pylori)	70
☐	L4	L2 same (helicobacter or pylori)	15
☐	L5	outer near membrane near (polypeptide or peptide or protein)	3608
☐	L6	L5 and (pylori or pyloridis or pylon or pylorum or pyloris or helicobacter or heliobacter)	534
☐	L7	L5.clm. and (pylori or pyloridis or pylon or pylorum or pyloris or helicobacter or heliobacter).clm.	11
☐	L8	L7 not l1	11
☐	L9	L8 not l4	10
☐	L10	bab or baba or bab-a or baba1 or baba2 or bab-a or babb or bab-b or babc or bab-c or bab-a1 or bab-a2	35558
☐	L11	L10 and (helicobacter or pylori or pyroli or pyloridis or pylorid or pyloridis or hpylori)	143
☐	L12	L10.clm. and (helicobacter or pylori or pyroli or pyloridis or pylorid or pyloridis or hpylori)	5
☐	L13	73.clm. or 74.clm. or 75.clm. or 76.clm.	100680
☐	L14	L13 same (heliobacter or helicobacter or pylori)	31

END OF SEARCH HISTORY

DOCUMENT-IDENTIFIER: US 20050009037 A1
TITLE: Helicobacter bizzozeronii outer membrane protein encoding gene and its use in diagnostic and treatment methods

Summary of Invention Paragraph:
[0007] Several families of outer-membrane protein (Omp) have been identified in H. pylori (Alm et al., "Comparative Genomics of Helicobacter pylori: Analysis of the Outer Membrane Protein Families," Infect. Immun. 68:4155-4168 (2000)). One major family (Hop) contains thirty-two homologous members, some of which are functional as porins (Peck et al., "Characterization of Four Members of a Multigene Family Encoding Outer Membrane Proteins of Helicobacter pylori and Their Potential for Vaccination," Microbes Infect. 3:171-179 (2001)). Some outer-membrane proteins not in paralogous families have been also identified. Among them are small-molecular-mass bacterial peptidoglycan-associated lipoproteins (PAL) such as Lpp20 and Omp22 (Kostrzynska et al., "Molecular Characterization of a Conserved 20-Kilodalton Membrane-Associated Lipoprotein Antigen of Helicobacter pylori," J. Bacteriol. 176:5938-5948 (1994); Kim et al., "Cloning and Characterization of a 22 kDa Outer-Membrane Protein (Omp22) from Helicobacter pylori," Mol. Cells 10:633-641 (2000)).

WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Thursday, February 23, 2006

Hide?	Set Name	Query	Hit Count
		DB=USPT; PLUR=YES; OP=OR	
☐	L1	human.clm. same colostrum.clm.	18
☐	L2	siga.clm. or s-iga.clm.	6
☐	L3	L2 and colostrum	0
☐	L4	secretory near3 iga	426
☐	L5	L4 and colostrum	68
☐	L6	l5 and (man or human or sapian or sapien)	67
☐	L7	l5 and ((man or human or sapian or sapien) same colostrum)	38

END OF SEARCH HISTORY

```
CLUSTAL W (1.81) multiple sequence alignment

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tr|Q9ZMK5|Q9ZMK5_HELPJ             MKKTILLSLMVSSLFAENDGVYMSVGYQIGEAAQMVKNTGEIQKVSNAY
                                                *:**.* ************

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- ☐ tr Q9ZKV2 _HELPJ Outer membrane protein-adhesin [babA] [Helicobac
- ☐ tr O25840 _HELPY Outer membrane protein (Omp28) [HP1243] [Helicob
- ☐ tr O25556 _HELPY Outer membrane protein (Omp19) [HP0896] [Helicob
- ☐ tr O25086 _HELPY Outer membrane protein (Omp9) [HP0317] [Helicoba
- ☐ tr Q8GNW2 _HELPY BabB (Fragment) [babB] [Helicobacter pylori (Cam
- ☐ tr Q8GNW1 _HELPY BabB (Fragment) [babB] [Helicobacter pylori (Cam
- ☐ tr Q8GNW0 _HELPY BabB (Fragment) [babB] [Helicobacter pylori (Cam
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- ☐ tr Q8GNV7 _HELPY BabB (Fragment) [babB] [Helicobacter pylori (Cam
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- ☐ tr Q7WV99 _HELPY Adhesin-binding fucosylated histo-blood group an
- ☐ tr Q7WV84 _HELPY Adhesin-binding fucosylated histo-blood group an
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- ☐ tr Q7WV69 _HELPY Adhesin-binding fucosylated histo-blood group an
- ☐ tr Q6T8D5 _HELPY BabB (BabB2) [Helicobacter pylori (Campylobacter
- ☐ tr Q6T8D3 _HELPY BabA [Helicobacter pylori (Campylobacter pylori)
- ☐ tr Q5Q1P2 _HELPY BabB/BabA1 fusion protein 1 (Fragment) [babB/bab
- ☐ tr Q5Q1P1 _HELPY BabB/BabA1 fusion protein 2 (Fragment) [babB/bab
- ☐ tr O52269 _HELPY Adhesin binding fucosylated histo-blood group an
- ☐ tr O51811 _HELPY Adhesin-binding fucosylated histo-blood group an
- ☐ tr Q6U2C5 _HELPY Adhesin-binding fucosylated histo-blood group (F
- ☐ tr Q5G5J5 _HELPY Adhesin-binding fucosylated histo-blood group (F
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- ☐ tr Q8GNW6 _HELPY BabB (Fragment) [babB] [Helicobacter pylori (Cam
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- ☐ tr Q8GNW4 _HELPY BabB (Fragment) [babB] [Helicobacter pylori (Cam
- ☐ tr Q8GNW3 _HELPY BabB (Fragment) [babB] [Helicobacter pylori (Cam

- [ ] tr Q7WVA5 _HELPY Adhesin-binding fucosylated histo-blood group an
- [ ] tr Q7WVA0 _HELPY Adhesin-binding fucosylated histo-blood group an
- [ ] tr Q7WV97 _HELPY Adhesin-binding fucosylated histo-blood group an
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- [ ] tr Q5G5J6 _HELPY Adhesin-binding fucosylated histo-blood group (F
- [ ] tr Q8GNX6 _HELPY BabB (Fragment) [babB] [Helicobacter pylori (Cam
- [ ] tr Q7WV98 _HELPY Adhesin-binding fucosylated histo-blood group an
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- [ ] tr Q6U2D4 _HELPY Adhesin-binding fucosylated histo-blood group (F
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- [ ] tr Q9Z390 _HELPJ Putative Outer membrane protein [jhp_0212] [Heli
- [ ] tr Q9ZN51 _HELPJ Putative Outer membrane protein [jhp_0007] [Heli
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- [ ] tr Q9X747 _HELPY HopZ protein precursor [hopZ] [Helicobacter pylo

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- ☐ tr Q7WV77 _HELPY Adhesin-binding fucosylated histo-blood group an
- ☐ tr Q7WV73 _HELPY Adhesin-binding fucosylated histo-blood group an
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- ☐ tr O24870 _HELPY Outer membrane protein (Omp2) [HP0025] [Helicoba

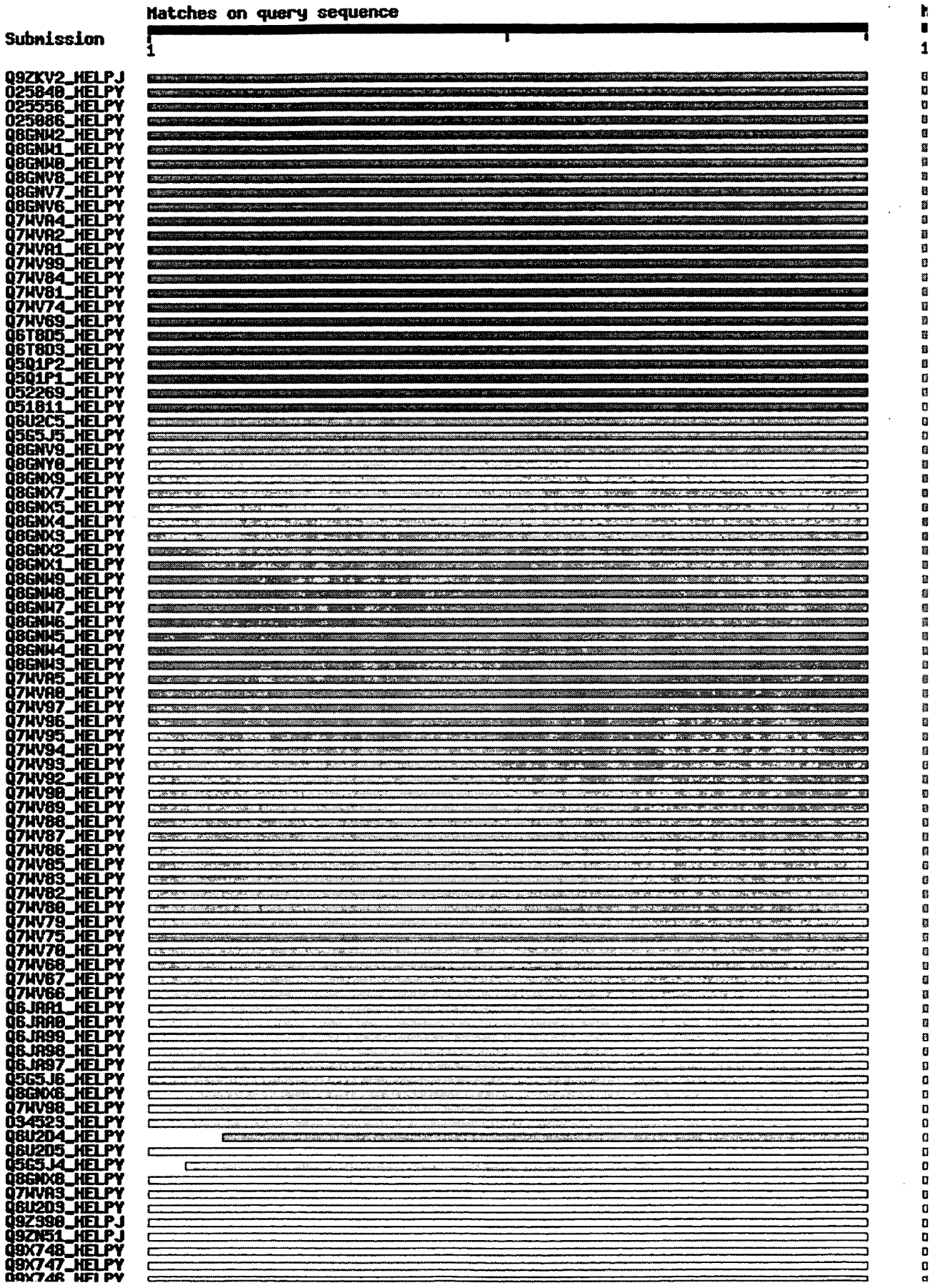
Graphical overview of the alignments

Click here to resubmit your query after masking regions matching PROSITE profiles or Pfam HMMs

(? Help) (use ScanProsite for more details about PROSITE matches)

Profile hits

Pfam hits


Matches on query sequence
Submission
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Q9ZN51_HELPJ
Q9X748_HELPY
Q9X747_HELPY

☐ 1: FEMS Immunol Med Microbiol. 1994 Jun;9(1):15-21. Related Articles, Links

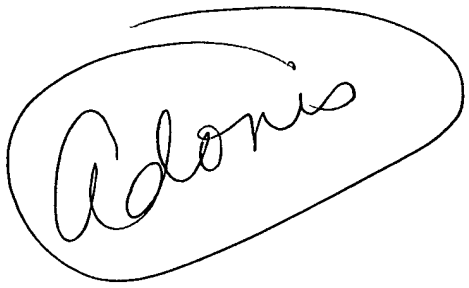
Detection of microbial surface antigens that bind Lewis(a) antigen.

Essery SD, Weir DM, James VS, Blackwell CC, Saadi AT, Busuttil A, Tzanakaki G.

Department of Medical Microbiology, University Medical School, University of Edinburgh, UK.

There is evidence that the Lewis(a) blood group antigen is one of the receptors for a number of potentially pathogenic microorganisms. To determine how widely distributed the microbial adhesins are that bind this antigen, anti-idiotypic antibodies produced against monoclonal anti-Lewis(a) were used in coagglutination assays to screen a variety of species. The following were agglutinated: 7/7 strains of Staphylococcus aureus; 10/19 (53%) strains of Neisseria meningitidis; 8/13 (62%) strains of Haemophilus influenzae; 1/3 strains of Helicobacter pylori; 1/2 strains of Neisseria gonorrhoeae; 1/2 strains of Candida albicans. The application of the anti-idiotypic antibodies to studies of host cell receptors, isolation of adhesins and development of new epidemiological typing reagents is discussed.

PMID: 7920460 [PubMed - indexed for MEDLINE]



Gastroenterology. 1997 Apr;112(4):1179-87. Related Articles, Links

Gastroenterology

Isolation of a cell surface component of Helicobacter pylori that binds H type 2, Lewis(a), and Lewis(b) antigens.

Alkout AM, Blackwell CC, Weir DM, Poxton IR, Elton RA, Luman W, Palmer K.

Department of Medical Microbiology, University of Edinburgh, Scotland.

BACKGROUND & AIMS: Individuals of blood group O and nonsecretors of ABO blood group antigens are more susceptible to peptic ulcers. The aim of this study was to determine if blood group antigens associated with group O or secretor status are epithelial cell receptors for Helicobacter pylori. METHODS: Bacterial binding and binding of monoclonal antibodies to H type 2, Lewis(a), and Lewis(b) to Kato III, buccal epithelial, and gastric mucosal cells were shown by flow cytometry. Bacterial outer membrane proteins eluted from H type 2, Lewis(a), or Lewis (b) were shown by polyacrylamide gel electrophoresis. RESULTS: Kato III and human epithelial cells bound each monoclonal antibody; O cells bound more anti-H type 2 ($P < 0.05$). Binding indices for H. pylori correlated with those for anti-H type 2 ($P < 0.005$) and anti-Lewis(b) ($P < 0.001$) but not anti-Lewis(a). A 61-kilodalton protein was eluted from H type 2, Lewis(a), or Lewis(b). CONCLUSIONS: Our results indicate that H type 2 is an important receptor for the 61-kilodalton bacterial adhesin, partly explaining increased susceptibility of individuals of blood group O to ulcers. Lewis(b) binds H. pylori more efficiently than Lewis(a). If these interactions occur in vivo, lack of Lewis(b) in mucosal fluids of nonsecretors may contribute to colonization by H. pylori.

PMID: 9098001 [PubMed - indexed for MEDLINE]

☐ 1: Gastroenterology. 1997 Apr;112(4):1179-87. Related Articles, Links

Gastroenterology

Isolation of a cell surface component of Helicobacter pylori that binds H type 2, Lewis(a), and Lewis(b) antigens.

Alkout AM, Blackwell CC, Weir DM, Poxton IR, Elton RA, Luman W, Palmer K.

Department of Medical Microbiology, University of Edinburgh, Scotland.

BACKGROUND & AIMS: Individuals of blood group O and nonsecretors of ABO blood group antigens are more susceptible to peptic ulcers. The aim of this study was to determine if blood group antigens associated with group O or secretor status are epithelial cell receptors for Helicobacter pylori. METHODS: Bacterial binding and binding of monoclonal antibodies to H type 2, Lewis(a), and Lewis(b) to Kato III, buccal epithelial, and gastric mucosal cells were shown by flow cytometry. Bacterial outer membrane proteins eluted from H type 2, Lewis(a), or Lewis (b) were shown by polyacrylamide gel electrophoresis. RESULTS: Kato III and human epithelial cells bound each monoclonal antibody; O cells bound more anti-H type 2 ($P < 0.05$). Binding indices for H. pylori correlated with those for anti-H type 2 ($P < 0.005$) and anti-Lewis(b) ($P < 0.001$) but not anti-Lewis(a). A 61-kilodalton protein was eluted from H type 2, Lewis(a), or Lewis(b). CONCLUSIONS: Our results indicate that H type 2 is an important receptor for the 61-kilodalton bacterial adhesin, partly explaining increased susceptibility of individuals of blood group O to ulcers. Lewis(b) binds H. pylori more efficiently than Lewis(a). If these interactions occur in vivo, lack of Lewis(b) in mucosal fluids of nonsecretors may contribute to colonization by H. pylori.

PMID: 9098001 [PubMed - indexed for MEDLINE]

All: 1 Review: 0

1: FEMS Immunol Med Microbiol. 1996 Dec 1;16(2):141-55.

ELSEVIER FULL-TEXT ARTICLE

Cell envelope characteristics of Helicobacter pylori: their role in adherence to mucosal surfaces and virulence.

Clyne M, Drumm B.

Department of Paediatrics, University College Dublin, Our Ladys Hospital for Sick Children, Crumlin, Ireland.

Helicobacter pylori colonises the gastric mucosa of humans and causes both antral gastritis and duodenal ulcer disease. Exactly how H. pylori causes disease is not known but several pathogenic determinants have been proposed for the organism. These include adhesins, cytotoxins and a range of different enzymes including urease, catalase and superoxide dismutase. Surface molecules of H. pylori such as flagella, lipopolysaccharide, the urease enzyme and outer membrane proteins are putative adhesin molecules. While phosphatidylethanolamine and the Lewis(b) blood group antigen have been proposed as receptor molecules for the organism the exact mechanism by which H. pylori adheres to the gastric mucosa has still to be identified. Characterisation of the adhesins of H. pylori could lead to the development of adhesin analogues for use in the inhibition of colonisation and improved therapy for ulcer disease. In vivo studies with isogenic mutants which are incapable of adhering to the gastric mucosa would greatly clarify the significance of adherence. Such mutants could possibly be useful as a vaccine against infection with wild-type organisms.

PMID: 8988394 [PubMed - indexed for MEDLINE]

☐ 1: APMIS. 1992 Oct;100(10):908-13. Related Articles, Links

Haemagglutination patterns of Helicobacter pylori. Frequency of sialic acid-specific and non-sialic acid-specific haemagglutinins.

Lelwala-Guruge J, Ljungh A, Wadstrom T.

Department of Medical Microbiology, University of Lund, Sweden.

Thirty-two Helicobacter pylori strains were screened for haemagglutination (HA) activity with erythrocytes of 11 different animal species. Twenty-three strains (72%) that agglutinated human erythrocytes exhibited a broad-spectrum HA profile. Human, guinea pig and bovine erythrocytes high in sialoglycoconjugates were strongly agglutinated by most strains. Except for two, seven strains (22%) that did not agglutinate human erythrocytes exhibited a narrow-spectrum HA profile, commonly not inhibitable by sialoglycoconjugates or N-acetylneuraminlactose (NANLac). Strains were classified into three major HA classes. HA of 10 strains (31%) in class I was inhibited by different combinations of NANLac, orosomucoid or fetuin, but not by asialofetuin, suggesting the presence of sialic acid-specific HAs probably recognizing NeuAc alpha-(2-3)- Gal isomer. Twelve strains (38%) in class II exhibited a different receptor specificity binding to different combinations of NANLac, orosomucoid and fetuin, as well as asialofetuin. No inhibition was observed with 10 strains (31%) in class III; thus, this receptor seems different from both the other classes. Of 21 strains (66%) in classes I and II, HA of 11 strains (34%) was inhibited with NANLac, 14 strains (44%) with orosomucoid and 15 strains (47%) with fetuin. The great heterogeneity observed in HA patterns indicates that the HAs of different strains may recognize a heterogeneous class of sialoglycoconjugates on the erythrocyte membrane.

PMID: 1445697 [PubMed - indexed for MEDLINE]

☐ 1: J Infect. 1992 May;24(3):263-7. Related Articles, Links

Adherence of Helicobacter pylori to HEp-2 cells.

Figueroa G, Portell DP, Soto V, Troncoso M.

Microbiology Unit, University of Chile, Santiago.

The adherence of 25 strains of Helicobacter pylori was evaluated in HEp-2 cells. These bacterial isolates, obtained from Chilean patients with gastric disorders, were also tested for haemagglutination of human red blood cells. Adherence of HEp-2 cells was expressed as a common property of all strains, irrespective of whether the cultures were grown on semi-solid or in liquid media. Previous reports that haemagglutinating activity was present in cells grown only on semi-solid media were confirmed. Adherence to HEp-2 cells was suppressed when bacterial cells were pretreated with homologous or heterologous whole human serum, containing specific antibodies of H. pylori. Adherence remained unaltered when bacterial cells were similarly treated with normal serum lacking specific antibodies. These observations imply that adhesions are expressed in vivo and suggest that an adherence mechanism, not depending on the expression of specific haemagglutinin antigen, operates for H. pylori.

PMID: 1602147 [PubMed - indexed for MEDLINE]

☐ 1: FEMS Immunol Med Microbiol. 1995 Feb;10(3-4):307-16. Related Articles, Links

Attachment, ingestion and intracellular killing of Helicobacter pylori by human peripheral blood mononuclear leukocytes and mouse peritoneal inflammatory macrophages.

Chmiela M, Paziak-Domanska B, Wadstrom T.

Department of Medical Microbiology, University of Lund, Sweden.

The different steps of phagocytosis, attachment, ingestion and intracellular killing of cells of Helicobacter pylori strain 17874 (expressing sialic acid-specific haemagglutinin) and cells of H. pylori strain 17875 (expressing non-sialic acid-specific haemagglutinin) have been studied. More cells of sialopositive H. pylori strain 17874 have been found attached to human peripheral blood mononuclear leukocytes (PBM) and mouse peritoneal inflammatory macrophages (PIM) than cells of sialonegative H. pylori strain 17875. Binding of cells of H. pylori strain 17874 has been significantly inhibited by treatment of phagocytes with neuraminidase. Inhibition of adhesion of these bacteria preincubated with foetuin to normal phagocytic cells has also been found. Well adhering cells of H. pylori strain 17874 were more resistant to killing mechanisms of human PBM and mouse PIM than cells of strain 17875. Good, probably sialic acid-specific haemagglutinin dependent, adhesion of H. pylori bacteria to phagocytes can be considered as an important virulence factor which facilitates the pathogen to avoid the defence mechanisms.

PMID: 7773248 [PubMed - indexed for MEDLINE]

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L1: Entry 11 of 15 File: USPT Jun 25, 2002

US-PAT-NO: 6410719
DOCUMENT-IDENTIFIER: US 6410719 B1

TITLE: Blood group antigen binding protein and corresponding agents

DATE-ISSUED: June 25, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Boren; Thomas	S-906 28 Ume.ang.			SE
Arnqvist; Anna	Ume.ang.			SE
Normark; Staffan	Stockholm			SE
Ilver; Dag	Ume.ang.			SE

US-CL-CURRENT: 536/23.7; 536/23.1

CLAIMS:

What is claimed is:

1. An isolated recombinant DNA from Helicobacter pylori encoding an adhesin protein, wherein said protein specifically binds to the fucosylated blood group antigens Lewis .sup.b and H-1, comprising the nucleotide acid sequence of SEQ ID NO:1.

2. A vector comprising an isolated recombinant DNA from Helicobacter pylori encoding an adhesin protein, wherein said protein specifically binds to the fucosylated blood group antigens Lewis.sup.b and H-1, comprising the nucleotide sequence of SEQ ID NO:1.

3. An isolated recombinant DNA from Helicobacter pylori encoding an adhesion protein, wherein said protein specifically binds to the fucosylated blood group antigens Lewis.sup.b and H-1, comprising the open reading frame of the nucleotide sequence of SEQ ID NO:1.

4. A vector comprising an isolated recombinant DNA from Helicobacter pyroli encoding an adhesin protein, wherein said protein specifically binds to the fucosylated blood group antigens Lewis.sup.b and H-1, comprising the open reading frame of the nucleotide sequence of SEQ ID NO:1.

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L1: Entry 9 of 15 File: USPT Mar 23, 2004

US-PAT-NO: 6709656
DOCUMENT-IDENTIFIER: US 6709656 B1

TITLE: Helicobacter pylori adhesin binding group antigen

DATE-ISSUED: March 23, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Boren; Thomas	Umea			SE
Arnqvist; Anna	21 Umea			SE
Hammarstrom; Lennart	86 Huddinge			SE
Normark; Staffan	41 Stockholm			SE
Ilver; Dag	35 Umea			SE

US-CL-CURRENT: 424/190.1; 424/184.1, 424/234.1, 435/252.1, 435/7.32, 514/25, 530/350

CLAIMS:

What is claimed is:

1. An isolated and purified bacterial blood group anitgen binding adhesin protein (BabA) from Helicobacter pylori species, wherein said protein binds specifically to fucosylated Lewis.sup.b type I and H-1 blood group anitgen-glycoconjugates and, wherein said protein contains less than 20% bacterial protein impurities, has a molecular weight in the interval of 73 to 75 kDa as determined by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE), and is not a HopA, HopB, HopC, HopD, or HopE protein.

2. The adhesion protein of claim 1, wherein the SEQ ID NO:5

EDDGFYTSVGYQIGEAAQMV

is in an amino terminal position.

3. The adhesion protein of claim 1, wherein the protein has a molecular weight of about 73.5 kDa, as determined by SDS-PAGE.

4. The adhesion protein of claim 3, wherein said Helicobacter pylori species is Helicobacter pylori strain CCUG 17875.

5. An immunogenic composition comprising an adhesion protein according to any one of claim 1, 2, 3, or 4, optionally together with pharmaceutically acceptable excipients.